



Nocturnal release of leukocyte-derived microparticles in males with obstructive sleep apnoea

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Résumé en anglais	<p>Multiple pathophysiological mechanisms have been proposed to contribute to the increased cardiovascular morbidity in obstructive sleep apnoea (OSA), including autonomic dysfunction, inflammation, oxidative stress and endothelial dysfunction 1. Microparticles (MPs) are small membrane vesicles that are shed from circulating cells or from the components of the vessel wall in response to activation and apoptosis. There is growing evidence in support of a potential role of MPs in the field of cardiovascular diseases. Increased levels of MPs derived from various cell types are found in patients at risk of cardiovascular diseases 2. By modulating inflammation, coagulation, vasomotor reactivity and angiogenesis, MPs might directly contribute to cardiovascular diseases 2. Recent case-control studies suggest a potential involvement of MPs in OSA-associated cardiovascular morbidity 3-6. An increase in morning levels of MPs derived from activated leukocytes has been demonstrated in otherwise healthy male OSA patients with marked nocturnal desaturations 5. In vitro, nitric oxide (NO) production by endothelial cells incubated with MPs from OSA patients correlates negatively with circulating levels of activated leukocyte-derived MPs 5. Ex vivo, mice previously injected with MPs from OSA patients display endothelial dysfunction, reduced endothelial NO release and increased adhesion molecule expression 5.</p>
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